coagulation occurred in the blood stream some time before death. The actual tumor present was distributed throughout the bone marrow, with only a few clinically demonstrable lesions in the skull.

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DISCUSSION

ALVIN G. FOORD, M.D. (Huntington Memorial Hospital, Pasadena).—This interesting case represents a clinical and pathologic syndrome which I am sure is frequently missed by clinicians and pathologists because of its bizarre characteristics. Most, I believe, are signed out as chronic nephritis because of the renal insufficiency accompanying the disease. Others are called unexplained malignancy, bizarre anemias, etc. Too often the pathologist fails to examine the bones at autopsy, and misses the diagnosis completely. In the last few years we have picked up eight cases in Pasadena, simply by instructing our blood-counting technicians to be on the lookout for, and present to their chief, blood smears in any case which presents exaggerated rouleau formation. These are all investigated. Particularly is the middle-aged or older individual having the external appearance of malignancy with a moderate or marked anemia in which one cannot determine the origin of a malignant process, suspected as being a possible case of myelomatosis. Thorough blood chemical studies, including blood proteins, urinalysis for Bence-Jones protein, and x-ray examination are in order, and usually the case will be settled. However, as in two of our cases, there is often little or no destruction of bone, and it may be difficult to convince oneself and the radiologist that diffuse growth of myeloma is present in the same marrow spaces. The case presented demonstrates this beautifully. Bone marrow biopsy should clinch the diagnosis.

One should be careful in interpreting or trying to give too much significance to exaggerated rouleau formation in blood smears. The commonest cause, of course, is slow drying of the blood film, and hence all smears should be made and dried very quickly. The amount of rouleau formation will parallel the rapidity of sedimentation of liquid blood, and is largely dependent, as the sedimentation rate on the concentration of the globulins and fibrinogen in the blood. In all of our cases the sedimentation rate has been the fastest observed in any clinical condition, including the severe infections. If one did routine sedimentation rates he should seldom miss one of these cases. However, some cases, especially those with multiple large discrete lesions, are not associated with hyperproteinemia and hyperglobulinemia, nor are those which lose large amounts of protein in the urine. In fact, a few may have even less proteins than normal, due to excessive urinary loss.

The note, that the pathologist in this case observed some tinctorial similarity of the protein in the blood vessels to amyloid, brings up the remark of Magnus-Levy, who, in reviewing the cases of multiple myelomatosis with autopsies in the literature, stated that about half of the cases showed true amyloidosis. In a recent verbal communication, Wells stated that he believes amyloid is a globulin. Certainly Bence-Jones protein is a globulin, or a not far removed protein. Why this disease is accompanied by the marked changes in the blood proteins I cannot say, but the disease gives good argument for the belief that the blood proteins are formed in the bone marrow.

HARRY A. WYCKOFF, M.D. (Stanford University Hospital, San Francisco).—This paper by Schumacher, Williams and Coltrin, is seasonal in that it emphasizes the importance of a valuable diagnostic clue leading to the recognition of myeloma. It is probable that this sign is relatively often overlooked or disregarded. Fahraens has clearly demonstrated the relationship of autohemagglutination and increased sedimentation rate of the red cells, and the association of autohemagglutination with increase of protein content of the plasma; particularly the fibrinogen and globulin fraction has come to be fairly generally recognized.

Although hyperproteinemia occurs in conditions other than myeloma, it is still sufficiently uncommon to make it a decidedly useful diagnostic aid. That hyperproteinemia may perhaps occur in its most pronounced form in myeloma also helps to direct suspicion to this disease.

The fact that myeloma may exist without increase in plasma protein must be kept in mind in this connection. It also appears that plasma protein content shows considerable variation during the course of the disease. It should further be remembered that Bence-Jones proteinuria may commonly occur without hyperproteinemia.

THE HEART: THE PRESENT STATUS OF PHYSICAL EXAMINATION*

By GUY VAN SCOYOC, M.D.
Los Angeles

DISCUSSION *by John J. Sampson, M. D., San Francisco; Saxton T. Pope, Jr., M. D., San Francisco.*

WITH the advance in our knowledge of electrocardiography and roentgenography in relation to the examination of the heart, one wonders just what place the physical examination of this organ now occupies. It is the purpose of this paper to attempt to throw some light on the present status, as well as to eliminate in a way some of the measures that have become obsolete by virtue of modern methods.

PHYSICAL EXAMINATION OF PATIENT'S HEART IS IMPORTANT

At the outset let me state that in my opinion the physical examination is as important today as ever, and it is in defense of this time-honored procedure that I present this paper.

With proper background and training, this procedure reveals more than ever before. This has been made possible by the more exact, confirmatory knowledge brought about by the use of the x-ray and electrocardiograph. By checking our physical findings with these instruments in any given case, we are liable to make a more correct physical diagnosis on the next patient who comes under our observation.

VALUE OF THE NEW PRECISION INSTRUMENTS

Unfortunately, there has been some misunderstanding as to the value of the more or less new

* Read before the General Medicine Section of the California Medical Association at the sixty-fifth annual session, Coronado, May 25-28, 1936.

precision instruments. I think if we consider them only as aids to our examination, the same as we use the various other laboratory procedures, a more satisfactory diagnosis will be made. There has been a tendency to abandon the physical examination of the heart in favor of instruments, only to find that instruments fail us at the critical time, and that we have to revert to what we see and hear.

Before proceeding, let me state that it is impossible to cover such a large subject as I have selected in the time allotted; so, of necessity, only the high points will be touched upon and those in a very brief manner.

INSPECTION AND PALPATION

Inspection and palpation of the heart may fairly be considered together, palpation confirming inspection findings. This portion of the examination should be local and general.

General Survey. — For a cardiac examination the patient should be stripped to the waist and, in some instances, the feet and legs bared. The latter has been made more necessary by the recent advances in our knowledge of peripheral vascular disease. If thermal changes with discoloration are found in the feet and hands, thrombi-angiitis obliterans, endarteritis obliterans, or Raynaud's disease should be considered. We should also look for edema, local or general, anasarca, character of the skin, petechial hemorrhages of subacute bacterial endocarditis, pallor, cyanosis, and dyspnea. Heart cases generally show dyspnea, especially in the presence of pulmonary edema or hydrothorax. If ambulatory, they show this conscious, increased respiration in varying grades with arteriosclerotic heart disease.

Dyspnea and cyanosis are closely related and frequently found together in valvular heart disease. Likewise, they may occur from extracardiac causes, such as compression of pulmonary veins, mediastinal tumor, aneurysm, coal-tar products, pneumonia, phthisis, edema of lungs, and pneumothorax. When cyanosis is out of proportion to the dyspnea, some of these exocardial conditions are liable to be the seat of the trouble.[1]

Inspection of the face is important. The blending of pallor and cyanosis may manifest itself in long decompensated heart disease, while aortic disease may show pallor alone. Mitral stenosis may give various tinges of yellow, while extreme cyanosis in the young adult almost invariably points to pulmonary stenosis. Edema of the face is generally of a cardiac nature, and the lips should be examined for capillary pulse of aortic insufficiency. If the pupils are not symmetrical and round, and provided injury and iritis are not a factor, a careful examination of the cardiac base is warranted, with syphilis in mind. Arcus senilis may be a clue to arteriosclerosis. Clubbing of fingers may be either pulmonary or cardiac.

If the veins of the neck or thorax are distended and tortuous, some intrathoracic compression is present that interferes with the return flow of the blood to the right heart. This may be a mediastinal tumor, a hypertrophied heart, or an aneurysm.

TABLE 1.—*Follow-Up Findings in 100 Cases of Apical Systolic Murmurs*

No murmurs	8	per cent
Uncomplicated systolic murmur	60	
Advanced cardiac disease	30	
Mitral stenosis		17 per cent
Aortic insufficiency		9
Combined		4
Deaths	2	

Similarly, veins of the lower part of the chest may communicate with a caput medusae, lending evidence of portal stasis and inferior vena cava engorgement.

Unilateral diminution of motion of a chest may be due to hydrothorax that accompanies cardiac decompensation. Occasionally the trachea will be shifted from its normal position by an aneurysm, but more frequently this occurs as the result of fibroid retraction of the lung from tuberculosis. If due to aneurysm, the characteristic tracheal tug may be present.

Local Survey.—After a general survey has been made, a local inspection of the apex and extra-apical surroundings is in order. If the apex is displaced to the right or left, it is due to hypertrophy or dilatation of the heart itself, or pressure or traction from without, which may be pleuropericardial adhesions, pleurisy with effusion, lung tumor, fibroid phthisis, syphilis of the lung, deformities either occupational or acquired, or pressure from below the diaphragm. The site of the heart's apex is somewhat influenced by age, structure of the thorax, and respiration.

Various extra-apical pulsations of the thorax and neck sometimes give a clue to the underlying pathology. A pulsation in the left anterior thorax, between the second and sixth rib, may mean a pulsating pleurisy or a fibroid retraction of the right border of the left lung; a pulsation on the posterior chest wall, below the angle of the left scapula, indicates a large pulmonary cavity filled with fluid, while a systolic retraction in this area means pleuropericardial adhesions. A systolic pulsation above the base of the heart, or to the right or left of the sternum in this area points to aneurysm of the aorta.

A systolic pulsation of the jugular vein, representing the pathologic positive venous pulse, is caused by direct regurgitation of blood into the right auricle, and is significant of tricuspid regurgitation. However, it may also happen if patent foramen ovale is present. Diastolic collapse of the jugular veins accompanies chronic adhesive pericarditis.

Thrills are merely palpable murmurs. The mechanism which is responsible for their production is the same as for murmurs, namely, the narrowing of an orifice through which the blood stream is propelled into a larger chamber of the heart, aorta, or pulmonary artery. In general a systolic thrill at the base indicates either aortic aneurysm, structural changes in the aortic and pulmonary valves, or exophthalmic goiter. A thrill, if located in the second interspace to the right of the sternum, is good evidence of aortic

TABLE 2.—*Substandard Issues of 1925 to 1931 Inclusive—Carried to Anniversary in 1933. Men and Women. Expected Dead by Company's Standard*

Mitral Regurgitation

Group	Hypertrophy	Entrants	Number Dead	Ratio	Expected Dead	Mortality Ratio
Class A	None	2,745	80	2.9%	47.35	169%
	Slight	1,173	35	3.0	18.92	185
	Moderate	419	25	6.0	6.76	370
Total		4,337	140	3.2%	73.03	192%
Class B	None	1,232	45	3.7%	26.09	172%
	Slight	375	41	10.9	6.48	633
	Moderate	175	20	11.4	3.02	662
Total		1,782	106	5.9%	35.59	298%
Class C	None	766	61	8.0%	16.29	374%
	Slight	289	25	8.7	5.67	441
	Moderate	128	14	10.9	2.53	553
Total		1,183	100	8.5%	24.49	408%

Mitral Regurgitation—Rheumatic Group

Group	Hypertrophy	Entrants	Number Dead	Ratio	Expected Dead	Mortality Ratio
Class A	None	1,113	33	3.0%	14.97	220%
	Slight	718	32	4.5	9.71	330
	Moderate	321	33	10.3	4.37	755
Total		2,152	98	4.6%	29.05	337%
Class B	None	596	30	5.0%	9.74	308%
	Slight	244	15	6.1	3.45	435
	Moderate	107	10	9.3	1.51	662
Total		947	55	5.8%	14.70	374%
Class C	None	120	11	9.2%	2.01	547%
	Slight	54	7	13.0	.71	986
	Moderate	35	5	14.3	.46	1,087
Total		209	23	11.0%	3.18	723%

Class "A," referred to in the table, comprises simple heart murmurs with no unfavorable history from any source and no other significant impairment.
Class "B" refers to a simple heart murmur, plus an unfavorable history from some outside source, such as inspection, and not found by original examiner.
Class "C" refers to heart murmurs plus one or more additional impairments of considerable significance, such as diabetes or renal disease.

stenosis, and this condition is not diagnosed without the presence of this characteristic palpable murmur.

Diastolic thrills are occasionally found with aortic and pulmonary regurgitant lesions, but the presystolic thrill at or just inside the apex is the one most frequently found, and is indicative of mitral stenosis. However, a similar thrill has been found to accompany a Flint murmur.

Systolic thrills at the apex may occur with mitral and tricuspid regurgitation. Pericardial friction fremitus may be elicited in the presence of acute fibrinous pericarditis, but this finding is generally more easily interpreted by the stethoscope.

PERCUSSION

Percussion is a valuable procedure that varies in direct proportion to the ability of the percussor. While it is a somewhat clumsy method of investigation, nevertheless, it is sufficiently accurate for practical purposes in a majority of cases, and until every physician can carry a fluoroscope to each patient, there is very little we can do except use it, as it has been estimated that far less than 50 per cent of heart cases have the advantage of the x-ray.[2] With a little careful practice, occasionally checking ourselves against fluoroscopic findings and six-foot x-ray plates, a great deal more confidence in the procedure can be obtained.

In the preparation of this paper I have tried to review most of the modern authors on this subject.[3,4] All believe percussion is satisfactory for practical clinical purposes, and that the waning stress placed upon it has been unjustified. My own experience, after twelve years of teaching physical diagnosis, is that in a majority of the cases valuable information can be obtained. For the right and left border the error is about one to two centimeters as against orthodiagraphy or the x-ray plate taken at a distance of six feet, the percussion marking being this amount in excess of the actual size of the heart.

The great vessels are hard to percuss. This is due to the angle of the gladiolus with the manubrium of the sternum at the second rib, resulting in the aorta and pulmonary artery being normally quite a distance from the posterior surface of the sternum, most of the sounds elicited being due to the vibration and resonating qualities of the sternum. However, if any marked increase occurs due to pathologic changes, percussion is possible. As percussion never represents the exact size of the heart, obscure cases should be given the benefit of the fluoroscope or x-ray. Of course, it is

not possible to percuss the heart accurately in the emphysematous chest or the obese subject; neither can much satisfaction be obtained by this method if pleurisy with effusion exists.

In the practice of percussion the sense of resistance to the pleximeter finger as we progress toward the cardiac borders is important, and contributes considerably to this method of examination.

In percussing the left border of the sternum, the most common cause of dullness in the second interspace is dilatation of the pulmonary artery from mitral stenosis, chronic emphysema, patent ductus arteriosis, pulmonary valve stenosis or aneurysm of the aortic arch. Dullness in the third interspace of more than two to four centimeters from the midsternum, varying with the size and build of the patient, means cardiac enlargement, displacement or pericardial effusion, or pulmonary artery enlargement. The maximum distance from the midsternal line at the left border should be from seven to nine centimeters, and is usually found in the fifth interspace to the left. Any deviation from this should lead to suspicion of hypertrophy, dilatation or displacement. The apex may be within normal limits and still have marked enlargement of a serious nature in the third interspace as a result of mitral stenosis.

Percussion of the right heart is more difficult and, fortunately, findings may be of less importance. Dullness in the first and second interspace to the right generally means aneurysm of the aorta, marked dilatation of the superior vena cava, or mediastinal tumor.

In the third and fourth interspaces to the right, it may be possible to percuss the right heart's border in small children or thin adults, but frequently no conclusions can be arrived at unless pathology is present, as the right border is pretty well covered with lung. If dullness is found, the following must be kept in mind: dextrocardia, dilated right auricle, cardiac hypertrophy, aneurysm, pericardial effusion, or displacement from some extracardial cause. It is not possible to distinguish the great vessels from the base of the heart by percussion, or the lower border which is closely apposed to the liver, in which instance the cardiac dullness blends with liver flatness.

AUSCULTATION

For convenience of description, heart sounds are studied from the standpoint of intensity, pitch, quality, and duration. Intensity of the various cardiac sounds varies in normal as well as in pathologic individuals. Thickness of the chest wall, emphysema, certain deformities, and cardiac dilatation diminish the normal intensity. Both sounds are accentuated, following excitement and ingestion of stimulants, acute febrile disease and exophthalmic goiter, while apparent accentuation may be noted in thin individuals or in fibroid retractions of the right sternal border of the left lung.

The pulmonic second sound is normally louder than the aortic in children, and is accentuated in any condition that raises the pressure in the lesser circulation, such as pneumonia, emphysema, regurgitant and stenotic lesions of the mitral and aortic valves, while diminution of the intensity of the pulmonary sound is indicative of failure of the right ventricle. Accentuation of the aortic sound indicates hypertension in the greater circulation, arteriosclerosis, chronic nephritis, uremia and apoplexy, and is normally louder in the adult. Accentuation of the first sound may be due to hypertrophy of the left ventricle, exophthalmic goiter or during the course of acute fevers; whereas myocardial degeneration, dilatation, anemia, and wasting diseases result in diminution of the intensity of the first sound. Poor quality of the first sound in arteriosclerotic heart disease has been pointed out by Viko,[5] who found 69 per cent of a series of 159 cases showing this defect.

By reduplication of the heart sounds we mean a separation of the elements of either the first or second sound, with no disturbance in the rhythm. If the two elements of either sound come almost together, it is sometimes called a "split sound." If they are separated by an appreciable interval, they are said to be reduplicated or doubled. The phenomenon has been attributed to an unequal closure of the leaflets of the mitral and tricuspid valves during ventricular systole. Reduplication of the first sound is limited to the apex and may be found in arteriosclerosis, myocardial degeneration, mitral stenosis, and adhesive pericarditis. Reduplication of the second sound is attributed to an asynchronous closure of the aortic and pulmonary valves. Any condition that causes an alteration in the pressure in the greater or lesser circulation may produce this physical finding.

Gallop rhythm was first described by Potain. It may be divided into presystolic and diastolic, the presystolic being more important from a clinical standpoint. It is characterized by the occurrence of a third sound just prior to the first sound of the heart, and has been noted in association with hypertension in the greater circulation prior to failure of the left ventricle, coronary thrombosis, chronic renal disease, as well as being associated with mitral stenosis. The mechanism of the production is not clear.

Thompson and Levine,[6] in a study of eighty-nine cases, found the average duration of life to be ten months and twenty days after detection of gallop rhythm. They did not separate the condition into the various phases as "presystolic" and "diastolic," considering the subject under one heading of "diastolic gallop rhythm." Splitting reduplication and gallop rhythm are diagnosed by the stethoscope alone.

It is interesting to note, and extremely fortunate, that the most common disturbances of rhythm coming under the physician's observation can generally be diagnosed by physical means.

Premature contractions or extrasystoles are contractions arising in the heart outside the sino-auricular node; they are also known as ectopic beats. Their occurrence is common, their significance varied, and for the most part they can be diagnosed without instruments, though sometimes graphic means are necessary.

After we have satisfied ourselves of our diagnosis of this abnormality, the question of sig-

nificance arises. About 70 per cent of these premature beats are ventricular, the balance for the most part auricular. There may also be a rare prematurity arising at the node.

In a general way it may be briefly stated that, given a normal heart otherwise, extrasystoles are of no serious import. They may be due to reflex causes, tobacco, drugs, infections, etc., or they may accompany myocardial damage. Sometimes they are present when the patient is quiet and the pulse is slow, to disappear upon acceleration of the heart rate. These are usually of a benign type. Whereas, also, others become more pronounced on exercise, these generally point to some myocardial focus. The frequency with which the disturbance occurs is of some importance, as it may readily be seen that the cardiac output may be diminished if they are frequent and persistent. The future of the patient is not influenced by the premature contraction, but rather by the underlying cause.

Sinus arrhythmia is seen often in children to and including puberty. Frequently in connection with respiration, it has to do with changes in control of sino-auricular node, by influence of the vagus. Diagnosis is usually easy. The irregularity is of the whole beat, and the pulse and apex correspond. It clears on exercise. We may suspicion paroxysmal tachycardia and auricular flutter when the heart rate is around 150, but the diagnosis is made by the electrocardiogram. Auricular flutter is less frequently found than paroxysmal tachycardia. Neither are very common.

In the normal heart the impulse of contraction proceeds in an orderly manner from the pacemaker to the auriculoventricular node, resulting in a normal contraction of the auricle, thus expelling the blood into the ventricle. Under certain pathologic conditions this orderly sequence of affairs becomes altered, resulting in a twitching, quivering, and extreme disorder of the auricular musculature. This we know as auricular fibrillation. The ventricle now, instead of receiving its normal stimuli, receives stimuli that vary in strength, and periodicity. The ventricle now picks up as many of the impulses as the auriculoventricular conduction system can handle. If the auriculoventricular system is unimpaired, the ventricular rate is rapid; but instead of the ventricle being allowed to rest for the next beat, it responds as best it can to the various impulses from the auricle. This results in a series of strong and weak beats that are "irregularly irregular." The pulse presents a "pulse deficit," by which we mean there is a discrepancy between the apex rate and the radial pulse rate, some of the contractions not being sufficient to open the aortic valves. This type of irregularity most frequently is the aftermath of mitral stenosis and hypertensive heart disease. It also occurs in exophthalmic goiter, and can nearly always be diagnosed by physical means by the irregularity of the heart beat, pulse deficit, and the increase of the disturbance by exercise, in contrast to many of the extra systoles.

Of the disturbances in conduction only partial and complete heart-block concern us here. The partial may be diagnosed by the true intermission of the apex and pulse in an otherwise normal rhythm. Complete heart-block may be suspected if the pulse is forty or under, and is not changed by position or exercise.

Delayed conduction and bundle-branch block are determined by the electrocardiogram. Pulsus alternans may be detected by the blood-pressure cuff, but is best identified by the polygraph.

The cardiac murmurs present a large subject in themselves and are pretty well understood. There is some tendency to regard the apical systolic murmur with more suspicion than in the past. It may signify rheumatic involvement in the young, and sclerotic heart disease or hypertensive heart disease in the old. I can best illustrate my meaning by two slides that will be shown later, one taken from the mortality studies of the New York Life,[7] the other from observations of Fineberg and Steuer.[8]

The mitral presystolic or the aortic diastolic murmur when detected should be regarded as a serious omen, for sooner or later trouble will ensue. Systolic murmurs at the aortic area are generally of organic nature. The lesion may be a stenosis of the valves or a roughening of the proximal portion of the aorta. Pulmonary systolic murmurs are very common. They are generally blowing in character and localized at the pulmonic area. They may be regarded as physiologic. Rarely may we see the pulmonic systolic, stenotic murmur of congenital heart disease and patent ductus arteriosus. The cardiorespiratory murmur was not found very frequently in my experience. Tricuspid regurgitant murmurs are generally relative, while stenotic murmurs at this area are usually congenital. I do not believe a fixed function test is suitable for all cases. It is certain that the heart of an elderly person should not respond to exercise as well as one of a young athlete in training, so some response to activity in keeping with the patient's normal mode of life seems obvious. Function tests as applied to the cardiac examination may reveal evidence of valvular disease such as mitral stenosis, regurgitant mitral lesions, as well as an occasional faint aortic diastolic murmur. Likewise, definite accentuation of the pulmonic second sound or snapping of the first sound warrants careful examination for mitral disease.

In closing, may I remind you that the end-result of our examination is to determine what the heart is capable of doing and the expectancy we can place upon it.

510 West Sixth Street.

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DISCUSSION

JOHN J. SAMPSON, M. D. (490 Post Street, San Francisco).—Doctor Van Scoyoc has presented an excellent review of the physical signs associated with heart disease. If space permitted, history taking should have been included in this article. It is a wise clinician who says, "Give your patient a chance and he will tell you his diagnosis." The personal interview with one's patient, necessary in history taking, often establishes a diagnosis of heart disease as well as evaluates heart function.

History and physical findings should not be disregarded when conflicting with technical reports. Three forms of special apparatus are commonly used in cadiac examinations, namely, the electrocardiograph, the roentgen ray, and the spirometer or other means of estimating cardiac function. One may indicate how too much emphasis has been placed on these facilities. Several years ago the value of electrocardiography was established in three groups of one hundred patients each at the University of California Hospital, namely, those patients with myocardial damage, with arrhythmias, and with miscellaneous variations such as caused by congenital lesions, etc. About 80 per cent were found correctly diagnosed prior to taking an electrocardiogram in each group. The electrocardiogram may give both positive and negative false evidence. Thus angina pectoris may produce no abnormal electrocardiograph change in 10 per cent of cases, and electrocardiograms taken one-half hour after an acute coronary occlusion may be quite normal. One particular patient at the University of California Heart Clinic has a normal electrocardiogram in the presence of both stenosis and insufficiency of the mitral and aortic valves. On the contrary, a young football player with a normal heart had 12 millimeters T waves, and another athlete a marked slurring of the R waves in all leads.

It has been learned by experience that "serious" arrhythmias, *i. e.*, auricular fibrillation, can be transient disturbances in an otherwise normal heart. Electrocardiogram ventricular preponderance is often a matter of the shape of a chest, and not of unequal ventricular muscle enlargement.

Cardiac roentgenograms notoriously fail to show the exact anatomical state of a heart. Mitral insufficiency or intraventricular septal defect may show nothing, even on fluoroscopy, if there is little dynamic disturbance, and strange distortions of heart shape may be encountered by variations in the shape of the chest and the height of the diaphragm.

No functional test has yet been devised as valuable as the history of a patient's ability to carry on various everyday activities without dyspnea.

Little has been left by Doctor Van Scoyoc for discussion of actual signs, but a few common findings that are often misinterpreted may be mentioned. In palpation, a thrill is often felt with the apex impulse in normal, thin individuals, especially children. A rocking apex impulse generally indicates left ventricular hypertrophy. The presence of an apex impulse well medial to a left-border dullness suggests either pericardial thickening or fluid, or an aneurysm of the left ventricle. The apex impulse as well as the left-border percussion dullness may be displaced to the left of the mid-clavicular line in normal hearts that are rotated upward and to the left by scoliosis. This is a common source of error in diagnosis of cardiac enlargement, as is displacement of the diaphragm and entire mediastinum from other causes.

Cardiac percussion has been considered of doubtful value in recent years; however, if an individual establishes his correct criteria of heavy or light percussion for various borders of the heart by frequent comparisons with roentgenograms, the procedure has real value. Percussion of heart dullness is naturally of little value in obesity or pulmonary emphysema.

In auscultation of the heart it seems that examiners have been too restricted by habits developed from didactic teaching on the characteristic areas of sound references from the particular valves. From roentgenogram of calcified valves it is not known that the aortic valve is normally under the sternum, or even to its left, and may be quite low in the chest. Under enlargement of various chambers the position of this or other valves may be so displaced as to entirely change the position commonly assumed by sounds or murmurs. The channels of blood flow must be considered likewise. Thus, rarely does one encounter mitral insufficiency without the systolic murmur being heard in the left subscapular region where the left auricle encroaches on the lung, or aortic stenosis without a clear systolic murmur heard in the carotid and subclavian arteries.

The third sound is another sign that is difficult to evaluate. Its presence in diastole may be termed gallop rhythm at all times, but it only carries the grave implication commonly assigned to this term, in the presence of a failing left ventricle. Certainly, this does not apply to the normal third sound heard especially in children, or to the "opening snap" of the mitral valve in mitral stenosis.

A clicking third sound is occasionally heard in midsystole, and has no apparent clinical significance.

The humming, continuous systolic and diastolic murmurs heard over the lower jugular veins, especially in children, have received very little attention, although ably described and explained by Hope in 1836, and is a normal finding.

Because of its ready application, blood pressure determination now constitutes a part of routine physical examinations, although the quality of the radial pulse will give much of the information obtained from this procedure. It is now recognized as a source of general information on heart function and metabolism, as well as merely peripheral arteriolar and capillary resistance. The pulse pressure generally parallels the volume of systolic output when other circulatory dynamic influences remain constant and thus serves as an excellent index of the failure or recovery of myocardial function.

It is important to assemble the various signs and symptoms of heart disease presented by an individual patient into a working unity and to be sure that they form a rational picture of the disturbed circulatory state, in order properly to evaluate them individually. Thus, dependant edema, whereas a sign of heart failure rarely is due to this mechanism unless there is an obviously elevated venous pressure as noted from distended jugular veins. Even in the presence of valvulitis, edema is more likely due to some other cause, such as concealed varicose veins of the legs, when high venous pressure is absent. Many other examples of signs and symptoms misinterpreted can be given, but among the common ones are tachycardia, pulmonary râles, cyanosis, chest pain, and even dyspnea. Thus, no one piece of evidence is pathognomonic of heart disease, but must be fitted into the signs and symptoms complex to establish its diagnostic or functional importance.

SAXTON T. POPE, JR., M. D. (University of California Medical School, San Francisco).—Doctor Van Scoyoc is to be commended almost as much on his choice of subject as on his exposition of it.

In attempting to establish a medical diagnosis, we see, we hear, we feel. Sometimes we smell. With extreme rarity, we taste. Decision is rested on the convergent evidence of several of our five senses.

The evidence is affirmed, if possible, by added methods of so-called precision. For the heart, these are electrocardiography and fluoroscopy. One must be grateful to them for the elucidation which, under proper usage, they can offer. Still one must deplore the disproportionate share of credit which they receive for their work.

It is an interesting test of the physician's skill and information to let him attempt an estimate based on physical diagnosis alone; that is, with stethoscope and sphygnomanometer permitted as his only tools.

In particular, I should like to praise Doctor Van Scoyoc's discussion of clinical methods for the segregation of the various arrhythmias.

Certain minor and technical objections to the body of the text at various points would include the following:

1. His use of the term "apex," under "Local Survey." Has it not been conceded generally that what one sees

(if he sees it) is the point of maximum impulse—a ventricular spot adjacent to the heart's true anatomical apex?

2. The omission of pneumothorax and massive collapse from among those pathologic processes which may affect the position of the point of maximum impulse. The former may considerably displace that point from the affected side. The latter may almost equally draw it toward it.

3. The omission from the list (emphysema and obesity) of an important complication to accurate cardiac percussion—the female breast, when generous.

I find myself in hearty concurrence with his admission of the difficulties of right-sided cardiac percussion, his interpretation of the major signs of cardiac disease, and his reverence for detailed physical examination.

DEAF AND HARD-OF-HEARING CHILDREN: THEIR PHYSICAL AND MENTAL NEEDS*

By REXFORD HOOBLER, M.D.
Oakland

DISCUSSION *by Elwood A. Stevenson, Superintendent of School for the Deaf, Berkeley; J. C. Edwards, M.D., Berkeley.*

DURING the past fourteen years, I have been Director of Health at our State School for the Deaf in Berkeley. This covers a span of years necessary for the hard-of-hearing boy or girl to assimilate an education. I have followed many of these children from the time they entered school until their technical education was complete, and frequently during this time I stopped to consider if we were doing all we could to advance their physical and mental needs.

In reading medical literature discoursing on the physical and mental needs of the child of defective hearing, I found the subject dismissed with the statement: "The health problems of the deaf are handled in a special manner by those in charge of the deaf." So often have I met with this statement that I begin to think we do have special physical and mental problems, and that the best way to improve them is to share their knowledge with the pediatricians of the State. I ask that you be more conscious of the need of your assistance for their better health.

There is a very definite lack of confidence in the medical profession on the part of the parents of these children. I give the creation of false hope as the cause of this, whether it be our fault or that of poachers on the medical profession. You may supply the remedy.

On entry to the school, only about 20 per cent of the children are immunized to smallpox and diphtheria. According to late surveys, this is fairly high. Although immunity to pertussis has been in vogue for the past two or three years, it has not reached these children before their arrival at the school. There is need for greater insistence on immunity to these diseases.

SCHOOL STAFFS

Some of our city schools have added to their staffs supervisors of the conservation of speech, sight, and hearing. This should be encouraged in your school, as prevention is the better way to attack the problem. Parents are being notified of defects in speech, sight, and hearing. If a parent should bring such a notice to you, please make a careful examination, as it may save some child from becoming hard of hearing, deaf, or blind. An audiometer is used to test the hearing of the children in most cases, and is probably more accurate in determining diminished hearing than any instrument you have in your office. This work has just started, and will probably make rapid progress.

CLASSIFICATION OF HEARING DEFECTS

If we are to keep up with what the educators expect of us, we must better equip ourselves and our offices. We should know how to classify the types of hearing defects. We should know how to sort the hard of hearing from the deaf child. We should know of the benefits of hearing aids and their limitations. We should know of the American Society for the Hard of Hearing and where the nearest League for the Hard of Hearing is. They should know of the nearest school for deaf children and of the facilities for lip-reading instruction for hard of hearing day-school children in our public schools. We should know what to say to the parents of these children as to their psychology and education. We should follow up every case to assist in its proper placement and its proper psychological adjustment.

IMPORTANCE OF SIGHT CONSERVATION

The deaf and hard of hearing are educated through the eye, to a far greater extent than normal children. Since the eye does double work, it is very necessary to conserve the vision. Thirty per cent of those with defective hearing wear glasses. On entry to the school, 10 per cent of the children are in need of or wear glasses. After two to five years, 25 per cent are in this class, and finally 30 per cent find the need of corrected vision.

STAFF AT THE STATE SCHOOL FOR THE DEAF

Our staff consists of a specialist in eye, ear, nose, and throat, a dentist, and a pediatrician. Contrary to general conception, the State School for the Deaf in Berkeley is not a home, clinic, or asylum, where the deaf and hard of hearing are committed, but a free boarding school where children of regular school age, capable of learning by special methods—are guests of the State for nine months of the year. The other three months of the year they return to their homes in your locality and are referred to you to correct defects not considered emergencies. We do not see all the hard of hearing and deaf children of the State. Many of them attend special classes for hard of hearing or deaf as day pupils in the public schools of the larger cities of the State, and so our special health problems and methods become yours as well.

A greater number of the pupils at the State School for the Deaf in Berkeley are totally deaf. They are sent there because of the greater difficulty in instruction, as well as the different methods used than with the hard of hearing. The ratio is ap-

*Read before the Pediatric Section of the California Medical Association at the sixty-sixth annual session, Del Monte, May 2-6, 1937.